Changes in Heart Rate and R-Wave Amplitude with Posture

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Abstract

Background: Effect of gravity on heart rate and blood pressure are well documented but the effect of posture on R-wave amplitude has not been studied. Aim: To investigate the effect of posture on the heart rate (HR) and R-wave amplitude (RWA). Methods: The electrocardiograph (ECG) was recorded in 20 young subjects on two occasions. A 5 minute recording of resting ECG was taken with the subject adopting the following postural cycle: lying, sitting, standing, sitting and lying positions. Results: The standing HR was significantly higher than that in sitting and lying positions, but the RWA was significantly lower in standing compared to the sitting and lying postures. The HR significantly increased at the initial phase of postural changes, irrespective of the position. For example, the HR increased from lying to sitting (66.6±12.3 to 85.0±10.9 beat · min^{-1}) but also increased when changed from sitting to lying (70.6±10.6 to 85.2±8.7 beat · min^{-1}). Conclusion: Body posture has an effect on HR and RWA. The changes are probably related to orthostatic haemodynamic stress as well as changes in cardiac electrical axis. Whether the cause of the initial increase in the HR during postural change was attributed to skeletal muscle reflex or venous return requires further investigation.

Key Words: heart rate, R-wave amplitude, posture, venous return, cardiac axis

Introduction

Body position and postural changes determine a gravitational gradient acting upon the cardiovascular and pulmonary systems. Body positioning can affect oxygen transport particularly in the elderly, obese, and critically ill. Body position is a common intervention used in the management of patients with acute cardiopulmonary dysfunction (6). Heart rate affects ventricular function and the R-wave amplitude (RWA) reflects myocardial ischaemia (2,9,18) and left ventricular function (1). RWA is influenced by the angle of projection of the left ventricular electrical field in relation to the anterior thoracic surface (20), suggesting an association between RWA and cardiac axis. There are however few reports in the literature describing the relationship between RWA and postures adopted for clinical purposes.

The relationship between postural changes and cardiovascular response via the heart rate (HR) and blood pressure has been extensively studied (4,7,25, 26,27). Resting HR is faster in up-right postures such as standing and sitting, compared with the lying position (25,26,28,31). The mechanisms proposed for such differences have been related to baroreceptor stimulation as well as the regulation of cardiovascular hormones (15,25,31). Pump and colleagues recently suggested that the working mechanism of baroreceptor stimulation was through suppression of the release of a vasoconstrictor, arginine vasopressin (27).

Sudden onset of lower limbs exercise in an upright position is accompanied by an increase in venous return through femoral veins, whereas a sudden cessation of such exercise is accompanied by a decrease in venous return (10). Employing synchronized beat-to-beat continuous monitoring of the HR and RWA parameters for analysis, He and colleagues demonstrated a simultaneous change in

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the R-wave amplitude (RWA) with heart rate and suggested a possible relationship between RWA and venous return caused by muscle activity (10).

While the changes in HR in relation to posture have been reported (26-28), the relationship between HR and RWA during postural changes has not been studied. Understanding this relationship will enhance appreciation of the dynamic response of the cardiovascular regulatory centre as well as heart rate regulatory mechanisms. The aim of this study was to investigate the relationship between the HR and RWA in different vertical and horizontal body postures as well as changes taking place during the alteration in posture.

Materials and Methods

Subjects and Exercise Protocols

Twenty university students participated in the study. Invitation to participate in the study was through an advertisement on the student notice board. The nature of the study and experimental procedures were explained to interested students. Written consent was obtained prior to data collection. All subjects declared that they were free from known cardiovascular dysfunction. Ethics approval was obtained from the University Departmental Research Ethics Committee.

Each subject participated in two separate data recording sessions. In both sessions, disposable ECG electrodes were attached to the anterior chest wall of the subject. A CM5-lead system was adopted in the present studies (3, 11). The chest electrodes were attached 1 inch inferior and left to the left nipple, 1 inch inferior to the suprasternal notch and a ground electrode was attached to the right chest below the nipple. These electrodes were attached to a pre-amplifier (Nihon Kohden, Tokyo), with the amplified ECG signal being fed to a digital oscilloscope (VC-11, Nihon Kohden, Tokyo) and through an A/D converter (DI220, DataQ, Ohio) to a PC computer.

The ECG signal was sampled at 400Hz, a frequency at which the maximal amplitude of R-wave is detected. The HR was calculated using the reciprocal of the RR interval. The average value of the late half cycle of the diastole was used as the baseline to compute the RWA (10).

Each subject rested in the sitting posture for 5 minutes prior to baseline HR and non-invasive blood pressure recording with an automated system (Vitalmax 800, Pace Tech Inc, FL). Measurement of blood pressure with non-invasive devices potentially underestimates pressure in non-erect postures, therefore blood pressure was measured only in the sitting position, and only for the purpose of establishing a steady baseline. ECG data recording commenced when HR and blood pressure data between two consecutive 5-minute intervals varied by less than 1%. ECG was recorded for 5 minutes at each posture using the following positional sequence during the first data recording session: supine lying, sitting, standing, sitting, and supine lying. During a second session, ECG data was collected for 5 minutes, in random order, in each of the following horizontal positions: flat supine lying, left side lying and right side lying.

All subjects wore loose clothing and during recording, subjects were reminded to lie quietly on the plinth without talking or sleeping. After each 5-minute recording, the subjects were instructed to change to the next position within 5 seconds. After the first recording session was completed, subjects were allowed a 10-minute rest before commencing the second session.

All experimental procedures were conducted in the physiology laboratory, and the room temperature was controlled between 22°C and 24°C. All subjects completed data recording for both sessions within the same afternoon.

Statistics

A two-way repeated measures analysis of variance (ANOVA) of mean HR and RWA recorded during each 5 minute data collection period was used to evaluate the change in each variable over time. Paired *t*-test was used to analyse any difference in the HR and RWA before and immediately after a postural change. Pearson correlation was used to analyse any relationship between the HR and RWA. Results were considered significant at the 95% confidence level ($P < 0.05$).

Results

Twenty healthy subjects (10 males) volunteered to participate in the study. The mean age of these subjects was 21 years, body mass of 60.4±14.2 (SD) kg and height of 168.2±8.7 cm. Their mean systolic and diastolic blood pressures in the sitting position prior to ECG recording were 105.1±9.1 and 71.7±7.9 mmHg respectively.

HR & RWA in Different Postures

There was a significant increase in the HR when the posture changed from horizontal to vertical, being highest in standing ($P<0.001$). The HR decreased upon returning from standing to the lying position (Table 1). The mean resting HR in the lying, sitting, standing, then sitting and finally lying positions was 66.1±12.1; 72.1±11.2; 79.3±10.6; 72.4±10.7 and

Table 1. Mean HR and RWA (±SD) recorded in the different postures during protocol 1.

Positions	HR (bpm)	RWA (mV)
Lying	66.12±12.15	2.29±0.68
Sitting	72.09±11.17	2.21±0.57
Standing	79.29±10.56	2.08±0.60
Return to sitting	72.39±10.73	2.12±0.59
Return to lying	65.13±10.68	2.25±0.67
P valve (Repeated Measures ANOVA)	0.000	0.000

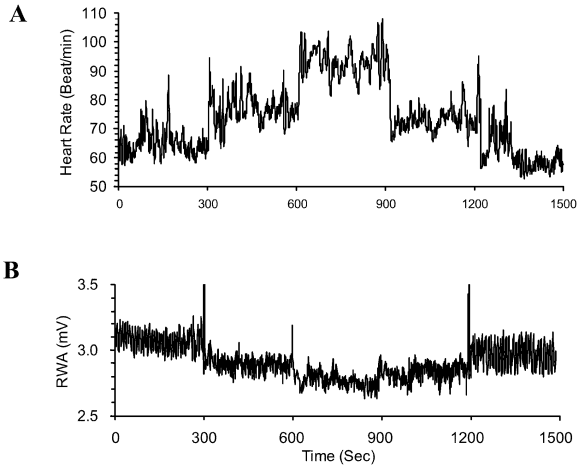

Fig. 1. Typical printouts of the time course for heart rate (HR) and R-wave amplitude (RWA) during Protocol 1: from supine to sitting, standing, then return to sitting, and finally supine. Each posture was maintained for 5 min. The printouts show the beat-to-beat information for the HR in A) and RWA in B).

finally 65.13±10.7 beat•min^{-1} respectively. The RWA on the other hand decreased from 2.29±0.68 mV in the lying position to 2.08±0.60 mV in standing. Though the change in the HR as well as RWA was significant between the horizontal and vertical positions, further contrast linear analysis demonstrated that the differences in these variables between the two vertical positions (sitting and standing) were not significant (P>0.05).

There were no changes in the HR in the horizontal positions, however the RWA decreased when the posture was changed from supine to the right side lying position (P=0.002, Linear Contrast Analysis) (Table 2). There was no significant difference in the RWA between supine and left side lying posture. The RWA in the left side lying position was higher than that in the right side lying position, but the difference did not reach statistical significance. Figures 1 and 2 illustrate the real time HR and RWA recorded at the different postures.

Table 2. Mean HR and RWA (±SD) recorded in the different postures during protocol 2.

Positions	HR (bpm)	RWA (mV)
Left side lying	64.58± 9.68	2.13±0.89
Supine lying	64.15± 9.46	2.27±0.78
Right side lying	65.31±10.43	1.92±0.61
P valve (Repeated Measures ANOVA)	0.23	0.011

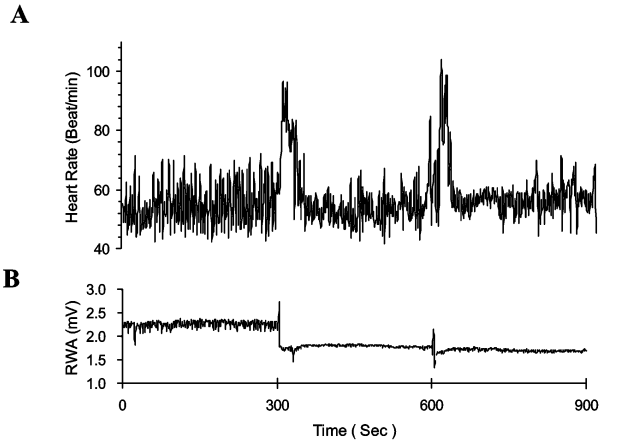

Fig. 2. Printouts of the time courses of HR and RWA during Protocol 2: from left side lying to supine and to right side lying postures. Data were obtained from the same subject and the printouts were synchronized.

Correlation between HR & RWA

In general, we observed a trend that the HR increased from supine to sitting, and further to standing posture; and likewise, a trend of decreasing RWA from lying to sitting and lowest in standing upright (Figure 1). However Pearson Correlation analysis showed that there was no statistical correlation in the changes of RWA in response to HR in the different positions (coefficient value ranged from 0.05 to 0.35 at the different positions, P>0.05).

HR and RWA at the Initial Phase of Position Changes

We observed from most of the subjects that the HR increased during the initial phase of all postural changes. Figure 1 illustrates an initial increase in HR when the subject changed from supine to sitting. A similar initial increase was also noted when the subject changed posture from sitting, back to supine position. Statistically, the HR increased significantly during the initial phase of position change and this occurred within the first 7 seconds after movement commenced. Table 3 illustrated that the mean HR increased from

Table 3. Mean HR and RWA recorded before and immediately after the change in positions during protocol 1.

Postural change	HR		RWA	
	Before	After	Before	After
Lying to sitting	66.61±12.30	85.00±10.93**	2.23±0.63	2.39±0.94
Sitting to standing	71.86±11.04	89.33±11.34**	2.09±0.59	2.21±0.63
Standing to sitting	76.03±10.74	86.09± 9.46**	2.09±0.57	2.27±0.68
Sitting to lying	70.63±10.57	85.20± 8.72**	2.17±0.63	2.58±0.92*

*P<0.05 and **P<0.001

Table 4. Mean HR and RWA recorded before and immediately after the change in positions during protocol 2.

Postural change	HR		RWA	
	Before	After	Before	After
Left to right	66.64± 3.04	79.13± 6.87*	1.38±0.21	1.12±0.44
Right to left	70.09± 7.01	93.81±11.45*	2.21±0.64	2.62±0.92
Supine to right	62.40±11.75	79.59±10.09**	2.36±0.65	2.22±0.72
Right to supine	64.94± 8.64	82.50± 6.26**	1.71±0.53	1.86±0.29
Left to supine	66.16±11.71	86.74± 9.07**	2.73±0.85	3.09±1.14
Supine to left	65.56± 8.50	80.09± 7.28**	1.76±0.39	1.89±0.90

*P<0.05 and **P<0.001

66.6±12.3 to 85.0±10.9 beat•min^{-1} (P<0.001) when the subjects changed from lying to sitting, and increased from 70.6±10.6 to 85.2±8.7 beat•min^{-1} (P<0.001) when the subject returned from sitting to lying. Similar results in increase in HR were found during the initial phase of change in the different horizontal positions (Table 4). RWA on the other hand, remained unchanged except when returning from the sitting to the lying position (P=0.04).

Discussion

Variation of heart rate is associated with postural change (23). In accord with previous reports, this study showed that HR was highest in standing compared to sitting or lying positions (12-14,25-27, 29,31). It is hypothesised that the increase in HR with standing follows a decease in venous return due to "venous pooling" in the lower limbs due to gravitational effects (4). The increase in peripheral venous volume is accompanied by an increase in both venous and arterial pressure in the lower extremities. The shift in blood volume from the central to the peripheral system induces a decrease in venous return and central venous pressure. The smaller the venous return, the smaller the end-diastolic and subsequent stroke volume. A reduction in venous return will lead to a reduced cardiac output, which in turn will lead to a reduction in baroreceptor stimulation in the aorta and carotid arteries (22). This reduction in baroreceptor firing results in decreased parasympathetic and increased sympathetic activity (7, 31). These two actions directly affect the cardiovascular centre in the medulla oblongata which increases the HR, the arteriolar and venous tones, and the cardiac contractility to compensate for the decrease in stroke volume and provide a cardiac output which can meet body demands. Upon returning the posture from sitting to lying, the increase in venous return increases the stroke volume through the Frank Starling mechanism, thus a lower heart rate is sufficient to maintain the cardiac output demanded by the body. Our finding is in accord with Pump and colleagues who reported a decrease in heart rate, with an increase in blood pressure, when the subject changed from a sitting to supine posture (26-28). It was these authors view that these blood pressure and heart rate changes were due to a posture-induced stimulation of the carotid and aortic pressure receptors. We did not measured blood pressure in this study because the changes in blood pressure in relation to changes in posture are well documented and the main focus of our study was the relationship between HR, RWA and postural change.

This study showed a decrease in RWA (from the resting posture) accompanied an increase in heart rate when moving from the lying to the sitting and standing positions. An increase in heart rate is accompanied by right cardiac axis deviation, which results in a less predominant RWA (30). This suggests that the decrease in RWA reported in our study could be a

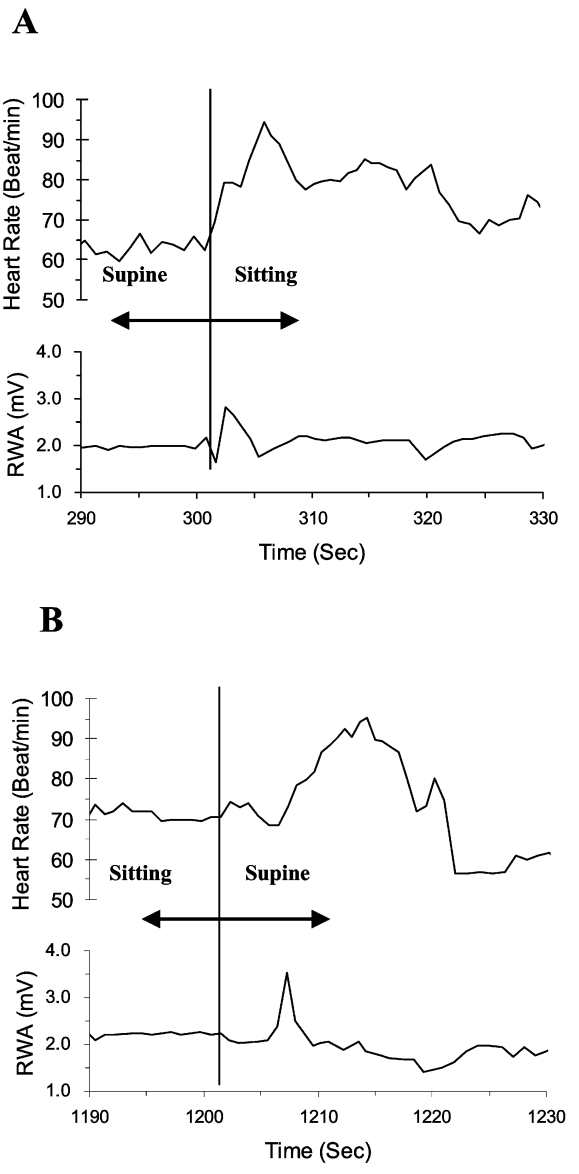

Fig. 3. Samples of the time courses of the HR and RWA during the initial phase of postural changes: A) from supine to sitting and B) from sitting to supine. Beat-to-beat information for a period of 40 sec is shown in each printout.

simple consequence of an increase in HR. This is supported by further analysis of our HR and RWA data illustrated in Figure 3, showing that the decrease in RWA was preceded by the increase in HR. Another explanation for the change of RWA in response to position variation is a change of cardiac axis with change in cardiac volume. Because of the reduction in central blood volume and reduced venous return when the posture is changed from lying to standing, the shape of the heart changes slightly, and this results in a change in the degree of projection of the left ventricular electrical field onto the anterior thoracic surface (20). An increase in R-wave amplitude is associated with a higher cardiac volume (18), and this could explain the increase in RWA in our study when the position was changed from sitting to lying. In summary, we postulate that the observed change in RWA resulted partly from the HR changes and partly due to the change of cardiac axis induced by a change in cardiac volume. The change in RWA was not significant between the two vertical positions (sitting and standing), and this is probably because the difference in venous return between these two positions was minimal.

When the subjects were horizontal, there was insufficient change in venous return to induce a cardiac response with supine positional change (28). RWA was much lower in the right side lying posture compared to supine. It is hypothesised that when one changes from the supine to the right side lying position, there is increased compression force from the chest wall against the heart (19), which could result in a change in the electrical impedance of cardiac muscle, thus reducing RWA. When turned to the left side however, we postulate that the change in the electrical impedance may be compensated for by the slight increase in venous return as the blood returns to the more dependent heart. This may explain why the difference in RWA was significant between supine lying and right side lying, but not between supine and left side lying positions.

Hemodynamics can be disturbed even by slight movements (8). In accord with previous findings (12-14,25-27,29,31), our results showed that irrespective of the postural change, there was a significant increase in HR within the first 7 seconds which commenced on initiation of any positional change, with each subject taking about 3 to 5 seconds to settle into a new position. Although muscle activity increases body oxygen consumption and energy expenditure, the temporal increase in HR associated with changing position suggests that it is unlikely to have resulted from the metabolic or hormonal consequences of increased muscle activity. We speculate that this increase was largely induced by the skeletal muscle reflex (5,16,17), although anticipation of exercise may also cause a rapid increase in heart rate (21).

The remarkable change in HR during the initial phase of postural change was not accompanied by a similar change in the RWA except when moving from sitting to lying position, suggesting that any variation in cardiac electrical activity was minimal. It is postulated that the significant change in RWA between sitting and lying postures reflects the greater increase in end-diastolic volume and cardiac electrical activity when a horizontal posture is adopted. Our previous study demonstrated an increase in RWA when walking exercise was suddenly initiated (10) and we postulated that this increase was due to a sudden increase in

venous return. On the other hand a decrease in the venous return is always associated with a slower HR, even during breath-hold diving (24).

This study demonstrated that body posture has an effect not only on the HR but also on the RWA. The HR was highest in the standing position compared to sitting and lying while RWA was lowest in standing. Increase in the HR occurred during the first 7 seconds following commencement of positional change. Variations in the RWA could be a consequence of the HR changes and the change of cardiac axis in response to posture. Changes in both parameters were likely related to venous return. Further studies are necessary to distinguish whether skeletal muscle reflex or venous return is the major determinant of change in these parameters. In summary, the study provides basic data for the effect of postural changes on cardiovascular responses in a young group of individuals with no known cardiovascular conditions. We propose that the effects of position-dependent cardiac responses may be exaggerated in populations that are older, overweight, pregnant, or suffering from cardiovascular dysfunction.

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